

Type	Drugs	Action	Uses	Adverse effects	Contraindication
HMG-COA REDUCTASE INHIBITORS "STATINS"	<ol style="list-style-type: none"> 1. Lovastatin 2. Atorvastatin 3. Fluvastatin 4. Pravastatin 5. Simvastatin 6. rosuvastatin 	<ul style="list-style-type: none"> • Inhibiting HMG CoA reductase • ↓↓ cholesterol (LDL & Triglyceride) • ↑↑ LDL Receptors • ↓↓↓LDL most effect 	<ol style="list-style-type: none"> 1. hypercholesterolemia by decrease LDL↓↓ 2. After AMI 3. Patients at high risk of coronary heart disease 	<ol style="list-style-type: none"> 1. Liver: Liver function disorders (elevated levels of <u>transaminase</u>) 2. Muscle: skeletal muscle weakness & pain common. Myopathy & even rhabdomyolysis occur rarely(<u>CPK levels</u>) 	<ol style="list-style-type: none"> 1. Pregnancy 2. Lactating women 3. Children and teenagers <ul style="list-style-type: none"> • Drug Interaction : Increase warfarin levels↑↑
Nicotinic acid	<ol style="list-style-type: none"> 1. Niacin (Vitamin B3) 	<ul style="list-style-type: none"> • Reduces lipolysis in adipose tissues • ↓↓ VLDL & LDL 	<ol style="list-style-type: none"> 1. Familial hyperlipidemias 	<ol style="list-style-type: none"> 1. Flushing ,pruritis most common prevented by taking aspirin prior to niacin 2. Liver dysfunction 3. Hyperglycaemia 4. Hyperuricaemia 5. Nausea & vomiting 	
Fibric acid derivatives	<ol style="list-style-type: none"> 1. Fenofibrate 2. Gemfibrozil 3. Clofibrate 	<ul style="list-style-type: none"> • Increase lipolysis of lipoprotein triglyceride via Lipoprotein lipase (LPL) • Reduces lipolysis in adipose tissues • ↓↓ VLDL & LDL • ↓↓↓↓triglyceride most effect 	<ol style="list-style-type: none"> 1. Hypertriglyceridemia 2. mixed hyperlipidemia 	<ol style="list-style-type: none"> 1. Mild GI disturbances (dyspepsia, abdominal pain) 2. Myositis, muscle weakness or tenderness, myopathy, rhabdomyolysis 3. Gallstones) increase biliary cholesterol excretion) 	<ol style="list-style-type: none"> 1. Pregnancy 2. lactation , 3. patients with severe hepatic & renal dysfunction & gallbladder disease
Bile acid sequestrants (resins)	<ol style="list-style-type: none"> 1. Cholestyramine 2. Colestipol 3. Colesevelam 	<ul style="list-style-type: none"> • Lowering the bile acid concentration by Resin/bile acid complex • ↓↓ cholesterol (LDL & Triglyceride) 	<ol style="list-style-type: none"> 1. drugs of choice in treating hyperlipidemias 2. Relieve pruritus caused by accumulation of bile acids in patients with biliary obstruction 	<ol style="list-style-type: none"> 1. Unpleasant taste & GI disturbances (constipation, diarrhea, flatulence, steatorrhea) 2. Interference with drug absorption as digoxin, thiazides, warfarin, aspirin 	
Cholesterol absorption inhibitors	<ol style="list-style-type: none"> 1. Ezetimibe 	<ul style="list-style-type: none"> • Selectively inhibits intestinal absorption of dietary and biliary cholesterol in the small intestine 	<ol style="list-style-type: none"> 1. Useful in hypercholesterolemia when a statin alone is inadequate 		

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Class I A Phase 0 (Na channel blockers)	Disopyramide	<ul style="list-style-type: none"> • ↑↑ Increase action potential duration 	<ol style="list-style-type: none"> 1. Orally 2. IV 	<ol style="list-style-type: none"> 1. PVC (premature ventricular contractions) 2. Ventricular arrhythmias (after AMI) 3. SVT of Wolf Parkinson White syndrome 	<ol style="list-style-type: none"> 1. Anti-muscarinic effects 2. Decrease blood pressure
Class I A Phase 0 (Na channel blockers)	Quinidine	<ul style="list-style-type: none"> • ↑↑ Increase action potential duration 		<ol style="list-style-type: none"> 1. Atrial fibrillation or flutter 2. Resistant SVT (Supraventricular Tachycardia) 3. Occasionally in ventricular tachycardia 	<ol style="list-style-type: none"> 1. Hypotension 2. heart failure
Class I A Phase 0 (Na channel blockers)	Procainamide	<ul style="list-style-type: none"> • ↑↑ Increase action potential duration 	<ol style="list-style-type: none"> 1. Initially by IV infusion then orally 	<ol style="list-style-type: none"> 1. Ventricular arrhythmias after AMI 	<ol style="list-style-type: none"> 1. Hypotension 2. prolonged therapy may cause drug-induced SLE
Class I B Phase 0 (Na channel blockers)	Lignocaine	<ul style="list-style-type: none"> • ↓↓ Decrease action potential duration 	<ol style="list-style-type: none"> 1. Only IV (infusion or injection) 	<ol style="list-style-type: none"> 1. PVC (premature ventricular contractions) 2. Ventricular tachycardia 3. Ventricular arrhythmias after AMI 	<ol style="list-style-type: none"> 1. Hypotension, 2. sleepiness , 3. confusion 4. convulsions
Class I B Phase 0 (Na channel blockers)	Phenytoin	<ul style="list-style-type: none"> • ↓↓ Decrease action potential duration 		<ol style="list-style-type: none"> 1. Digitalis-induced arrhythmias 	
Class I B Phase 0 (Na channel blockers)	Mexiletine	<ul style="list-style-type: none"> • ↓↓ Decrease action potential duration 	<ol style="list-style-type: none"> 1. orally 	<ol style="list-style-type: none"> 1. Ventricular arrhythmias after AMI 	<ol style="list-style-type: none"> 1. Hypotension 2. Tremor 3. Ataxia 4. dysarthria
Class I C Phase 0 (Na channel blockers)	Flecainide	<ul style="list-style-type: none"> • Negligible effects on action potential duration 		<ol style="list-style-type: none"> 1. PVC (premature ventricular contractions) 2. ventricular tachycardia 3. SVT (Supraventricular Tachycardia) 	

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Class II Phase 4 (Beta-blockers)	<ol style="list-style-type: none"> 1. Propranolol 2. Esmolol 			<ol style="list-style-type: none"> 1. APC (atrial premature contractions) 2. SVT (Supraventricular Tachycardia) 3. Atrial fibrillation 	
Class III Phase 1,2,3 (K channel blockers)	Amiodarone	<ul style="list-style-type: none"> • lengthen refractoriness • prolong action potential duration • Increases refractory period 	<ol style="list-style-type: none"> 1. Once daily orally 2. by injection 	<ol style="list-style-type: none"> 1. Atrial fibrillation 2. Ventricular tachycardia 3. SVT (Supraventricular Tachycardia) 4. WPWS arrhythmias 	<ol style="list-style-type: none"> 1. Corneal microdeposit (photophobia) 2. Photosensitivity 3. Thyroid disorders 4. Pneumonitis 5. pulmonary fibrosis 6. hepatitis
Class III Phase 1,2,3 (K channel blockers)	Bretylium	<ul style="list-style-type: none"> • lengthen refractoriness • prolong action potential duration • Increases refractory period 	<ol style="list-style-type: none"> 1. IV 	<ol style="list-style-type: none"> 1. In resistant ventricular arrhythmias after AMI like VF & VT 	
Class IV Phase 2 (Ca channel blockers)	Verapamil	<ul style="list-style-type: none"> • Direct –ve inotropic effects & -ve chronotropic effect • Blocking influx of calcium through L-type channels 		<ol style="list-style-type: none"> 1. SVT (Supraventricular Tachycardia) 2. Atrial fibrillation 	<ol style="list-style-type: none"> 1. Headache 2. Constipation, 3. Hypotension, 4. Bradycardia <ul style="list-style-type: none"> • Not used with beta-blockers • Contraindicated in heart failure and after AMI
Other	Adenosine	<ul style="list-style-type: none"> • slows & inhibits AV nodal conduction 	<ol style="list-style-type: none"> 1. IV 	<ol style="list-style-type: none"> 1. SVT (Supraventricular Tachycardia) 	<ol style="list-style-type: none"> 1. Bronchospasm(avoided in asthma) 2. flushing 3. chest pain
Other	Digoxin	<ul style="list-style-type: none"> • Inhibiting ATPase (Na-pump) in cardiac cells • Indirect –ve chronotropic effect through increasing vagus tone 	<ol style="list-style-type: none"> 1. Orally 2. IV 	<ol style="list-style-type: none"> 1. Atrial fibrillation 2. Arrhythmias as AF & SVT 3. Heart failure particularly when associated with arrhythmia likely AF <ul style="list-style-type: none"> • Smaller doses are used in: Elderly, renal disease, hypothyroidism, in the presence of hypokalemia 	<ol style="list-style-type: none"> 1. Digoxin toxicity include: <ul style="list-style-type: none"> • Cardiac effects: arrhythmias and heart block • GI effects: nausea and vomiting. • CNS effects: headache, confusion, nightmares, psychosis, coloured vision